Amendments to the Specification

Please substitute the following replacement paragraph(s) for the previously-pending versions of such paragraph(s). The replacement paragraph(s) are marked-up to show changes from the previously-pending versions thereof.

*** Replace paragraph [0001] as follows:

[0001] In patients with normal kidney function, calcium and ~~phosphorous~~ **phosphorus** balance is maintained through the interaction of parathyroid hormone (PTH) and calcitriol, an active metabolite of vitamin D. PTH provides a mechanism for controlling extracellular calcium and phosphate concentrations by regulating intestinal reabsorption, renal excretion, and exchange of these ions between the extracellular fluid and bone.

*** Replace paragraph [0003] as follows:

[0003] The retention of phosphorus as a result of the decreased ability of the diseased kidney to excrete the filtered phosphate leads to a decrease in serum free calcium, which in turn stimulates the secretion of more PTH. With each progressive reduction in kidney function, a new steady state is achieved in which serum phosphate is restored to normal at the expense of a sustained high level of PTH. The cycle is repeated as renal function declines until sustained and severe hyperparathyroidism is present; eventually the compensatory mechanism is not able to control the increasing serum ~~phosphorous~~ **phosphorus** levels. Once the glomerular filtration rate has decreased to <20% of normal, overt hyperphosphatemia becomes evident. In end-stage renal disease patients (where the compensatory mechanism mediated by PTH is no longer effective), the increase in plasma phosphate results not only from decreased excretion but also from continual high levels of PTH, which further exacerbates the problem by releasing calcium and phosphate from the bone.

*** Replace paragraph [0004] as follows:

[0004] The clinical manifestations of hyperphosphatemia are varied and have considerable mortality risks. Severe hyperphosphatemia can induce hypocalcemia, which aggravates the imbalance in PTH levels further by increasing the production of this hormone. Hyperphosphatemia inhibits renal synthesis of calcitriol, which causes an exacerbation of the hypocalcemia condition. The occurrence of severe hypocalcemia with tetany and ectopic calcifications is the most severe manifestation of hyperphosphatemia. Calcification may occur in the joints, soft tissues, lungs, kidney, and ~~conjuctiva~~ **conjunctiva**. Soft tissue calcification has been linked to cardiovascular risk, and cardiovascular disease is the cause of death in more than 45% of all dialysis patients. Renal osteodystrophy with effects on the bones and muscles is common in end stage renal disease (ESRD) patients, as well as severe pruritis. The high PTH level associated with developing and severe renal disease has indirect actions on the central and peripheral nervous system, and the myocardial tissues, creating further disorders such as hyperlipemia, muscle growth retardation, arteriosclerosis, bone loss, and immunodeficiency.

*** Replace paragraph [0045] as follows:

[0045] Other diseases that can be treated with the methods, compositions, and kits of the present invention include hypocalcemia, hyperparathyroidism, depressed renal synthesis of calcitriol, tetany due to hypocalcemia, renal insufficiency, and ectopic calcification in soft tissues including calcifications in joints, lungs, kidney, ~~conjuctiva~~ **conjunctiva**, and myocardial tissues. Also, the present invention can be used to treat ESRD and dialysis patients, including prophylactic treatment of any of the above.

[NO FURTHER ENTRIES THIS PAGE; REMARKS BEGIN ON THE NEXT PAGE]